APPENDIX A

APPENDIX A

Mr. Martoma Received No Material Non-Public Information

EVIDENCE	CITATION	TRANSCRIPT/EXHIBIT REFERENCE
(a) VASOGENIC EDEMA		
The information that Dr. Gilman and Dr. Ross allegedly shared concerning vasogenic edema was public.		
Elan and Wyeth publicly discussed vasogenic edema as a side effect of bapi in the presentation of the Phase I trial results on April 20, 2006.	DX 747, at 20 (Presentation of Phase I results given on April 20, 2006).	"All cases had reversible high signal on FLAIR sequences ?Consistent with vasogenic edema"
Dr. Enchi Liu testified that Elan and Wyeth publicly discussed vasogenic edema as a side effect of bapi in the presentation of the Phase I trial results on April 20, 2006.	Tr. at 954:25-955:17 (discussing DX 747, at 20).	25 Q. Can you turn to slide 20, please. We can actually help you 1 on that one, Ms. Liu, because it is up on the board. So, in 2 this presentation presented at this Geneva Springfield 3 symposium in Switzerland, Wyeth and Elan announced to the 4 public that they had seen MRI abnormalities in three to ten 5 patients at highest dose in study, right? 6 A. Yes. 7 Q. Do you see that? 8 A. Mmm-hmm. 9 Q. They went on to say that, "all cases had reversible high 10 signal on FLAIR. See consequences consistent with vasogenic 11 edema." Right? 12 A. Mmm-hmm. 13 Q. So, fair to say that back in the spring of 2006, Elan and 14 Wyeth had disclosed to the public that vasogenic edema was a 15 condition that they saw associated with the administering of 16 bapineuzumab in Phase I? 17 A. Yes.
Dr. Sidney Gilman testified that Elan and Wyeth	Tr. at 1587:12-1588:14.	12 My question is, isn't it a fact, Dr. Gilman, that you 13 were aware that vasogenic edema was reported as a side effect

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publicly discussed vasogenic edema as a side effect of bapi in the presentation of the Phase I trial results on April 20, 2006.		14 of bapineuzumab prior to December 2006? 15 A. Yes, I was aware of it before December of 2006. * * * 25 Q. But you're aware that the company disclosed it publicly to 1 the world by April 2006, when it disclosed publicly the results 2 of the Phase I study, right? 3 A. Yes. 4 Q. And you know that Phase I study was disclosed at a 5 conference that happened in Geneva, Switzerland, right? 6 A. Yes. 7 Q. Did you attend that conference? 8 A. I did not. 9 Q. And fair to say that that conference was a conference open 10 to the public, right? 11 A. Yes. 12 Q. And you know that when material is presented at a 13 conference like that, it becomes publicly available, right? 14 A. Yes.
Elan publicly discussed vasogenic edema as a side effect of bapi on a July 26, 2007, earnings call.	DX 172-A, at 11 (Transcript of July 26, 2007, Elan Q2 Earnings Call). *See also* DX 172 (Audio of July 26, 2007, Elan Q2 Earnings Call).	Corey Davis (Analyst, Natexis Bleichroeder): "And then the next question is can you share what you've learned about the potential for betagenic edema and how you'll be able to minimize the potential for that during the Phase III, and also once approved, my bigger question is would you anticipate the patients would have to be required to get regular MRI scans, given that edema is asymptomatic?" Lars Ekman (EVP, R&D, Elan Corporation, plc): "Right. It's important -- your last comment there, the betagenic edema is primarily a lab finding. We can see in the MRIs that basically we are very limited, if any clinical findings. That's one important point. The second point is that during the Phase II, in their relative patients that we have, we have been able to identify a time range when it occurs. We also are gaining knowledge of which patients could be at risk and how we should manage that risk, and obviously all of this would be reflected in the design of our Phase III trial."

EVIDENCE	CITATION	TRANSCRIPT/EXHIBIT REFERENCE
Elan publicly discussed vasogenic edema as a side effect of bapi on an October 25, 2007, earnings call.	DX 173-A, at 14 (Transcript of October 25, 2007, Elan Q3 Earnings Call). *See also* DX 173 (Audio of October 25, 2007, Elan Q3 Earnings Call).	Bill Tanner (Analyst, Lehring Swan): "Okay. Just on the dosing, you understand the APOE carrier, versus noncarrier, to sort of understand the rationale. Was the dosing then really arrived at more, or the differential dosing arrived at more from a safety perspective, trying to avoid the vasogenic edema? I'm guessing, what is known then about the relative efficacy and the two different types of patients. I don't know if you disclosed or it, or if you can remind us in the ongoing phase two, the percentage of APOE carriers?" Lars Ekman (EVP, R&D, Elan Corporation, plc): "We have not disclosed any efficacy in these two genome types. That's obviously, something that will fall off, as we know the totality of the data, and it would then be disclosed. We know that there is an abundance of vasogenic edema in the APOE four carriers, although the totality of the patients is low the relative percentage is higher in the APOE four carriers, and by having a lower dose in that patient category, we can significantly reduce this event. That has had an impact on the design."
Elan and Wyeth publicly discussed vasogenic edema as a side effect of bapi in the press release of the Phase II results on June 17, 2008.	GX 10, at 1-2 (Elan's and Wyeth's June 17, 2008, press release).	"As expected given the nature of the population studied, adverse events were very common in both placebo and bapineuzumab-treated patients. In non-carriers, the number of patients experiencing serious adverse events was similar between placebo and bapineuzumab-treated patients. In carriers, serious adverse events were more frequently observed in bapineuzumab-treated patients than in placebo patients. In addition, vasogenic edema was reported in the treated population with an increased frequency in carriers and at higher doses. No cases were reported in placebo patients. In the ongoing Phase 3 studies, carriers of the ApoE4 allele are being treated with a lower dose to minimize the risk of vasogenic edema. The Companies believe that the overall safety findings from this Phase 2 trial support their prior decision to move to Phase 3 studies."
Dr. Sidney Gilman testified that Elan and Wyeth publicly discussed vasogenic edema as a side effect of bapi in the press	Tr. at 1605:6-13.	6 Q. Now, you testified earlier that you were familiar with this 7 press release; isn't that right, Dr. Gilman? 8 A. Yes, but it's good to see it again. 9 Q. So is it fair to say that the dose depended frequency of 10 vasogenic edema in carriers of the ApoE4 gene was publicly

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release of the Phase II results on June 17, 2008.		11 disclosed information as of the time of this June 17, 2008 12 press release? 13 A. Yes.
Dr. Allison Hulme testified that Elan and Wyeth publicly discussed vasogenic edema as a side effect of bapi in the press release of the Phase II results on June 17, 2008.	Tr. at 364:1-365:6.	Q. The press release also talked about safety findings with 2 respect to the bapineuzumab trial, right? 3 A. That is correct, yes. 4 Q. If we could turn, I think it really flips over to the next 5 page where you see the next page where it says safety findings, 6 and then it goes on to talk about it. 7 One of the safety findings it talked about, Ms. Hulme, 8 was a condition called vasogenic edema. Is that right? 9 A. That is correct. * * * 19 Q. One of the these that was disclosed in the press release 20 was that vasogenic edema was observed in some certain patients, 21 but that the condition was treatable and would generally 22 resolve itself, and also that it was connected to how much dose 23 or how much of the bapineuzumab drug the person received, 24 correct? 25 A. In this press release we said that it was reported with an 1 increase frequency in the carrier population of the APOE4s and 2 at higher dose. 3 Q. In other words, saying for the people who had that gene, 4 they would have more frequency of this side effect and they 5 would get it more as the dose of the drug increased? 6 A. That is correct, yes.
Dr. Joel Ross testified that Elan and Wyeth publicly discussed vasogenic edema as a side effect of bapi in	Tr. at 747:11-14.	11 And you agree, Dr. Ross, that the public press release 12 on June 17, 2008 by Elan also announced the side effect of 13 brain swelling, or vasogenic edema, isn't that correct? 14 A. Yes

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the press release of the Phase II results on June 17, 2008.		
Dr. Sidney Gilman publicly stated to investors that vasogenic edema was a side effect of bapi during an April 15, 2008, GLG roundtable.	DX 759, at 1-2 (Rene Shen's notes from an April 15, 2008, GLG roundtable with Sid Gilman).	"Very interesting that the ApoE4+ means that you have more amyloid plaque and vasogenic edema is due to the efficacy of the drug potentially in removing the plaque from the vessel walls." * * * "There is a dose relationship where the higher the dose of AAB-001 that you give, the more likely you are to get vasogenic edema." * * * "The antibody has the ability itself to lift off the amyloid beta which likely leads the vasogenic edema of the AAB-001 at six weeks after immunization. Mostly after the first 2 shots, occasionally after 3-4 but rarely afterwards. There is more beta amyloid in the apo-E carrier group, so there is more vasogenic edema. Apo-E-4 status and dose. Greater prevalence of the VE as you get to higher doses. Removes it from the brain vessel wall. The N-terminal gets in there, and also the deposit beta amyloid." * * * "4,100 patients. The largest group is non-carrier. The carrier group will be 800 in each of two cohorts. The non-carrier is with the .5, 1, and 2 mg. [T]he carrier will get only .5mg so only one dose. He had a meeting with ELN/WYE, what happens if they miss the effective dose with .5mg. [I]t turned out to be a disagreement between the two companies. They want a safe drug. They are more concerned about vasogenic edema than losing some efficacy. Maybe they can add on an arm later after the P2. [T]hey may be underdosing. The P2 trial was permitted to be unblinded by the FDA. They were going to complete the trial anyway, so they were allowed to peak in order to power their study. They only had the .5 mg dose when they peaked. They have asked the P2 trial to be allowed as a P3 trial if it is 'dynamite.'"

EVIDENCE	CITATION	TRANSCRIPT/EXHIBIT REFERENCE
	Tr. 2502 at 18-20.	18 Q. Are these a copy of the notes that you took of a 19 consultation on April 15 at a round table given by Dr. Gilman? 20 A. Yes.
Dr. Sidney Gilman discussed vasogenic edema as a side effect of bapi during a consultation with Rene Shen on May 1, 2008.	DX 760, at 1 (Rene Shen's notes of a May 1, 2008, consultation with Dr. Gilman).	"Vasogenic edema – it is very benign. You just stop immunizing. The greatest risk factor is the A4 allele. They have a lot more AB in the brain."
Dr. Sidney Gilman discussed vasogenic edema as a side effect of bapi during a consultation with Rene Shen on June 17, 2008.	DX 761, at 1-2 (Rene Shen's notes of a June 17, 2008, consultation with Dr. Gilman).	"Vasogenic edema – there were cases at 1 mg. [T]here were at 1mg/kg 3-4 cases. 201 is the study that they are reporting on now. 202 is the study in Europe with PIB and PET. 3 1mg/kg cases. 10 at 2mg/kg. 1 at 0.15mg, that was an odd case that had a stroke, pre-existing cases. Of those cases the 2 non carriers in the 2 mg/kg group, so they were both at the highest dose. The carriers, none at .5mg. 1 at the .15mg dose, that was a stupid case. There is a one case in the open label that got it at .5mg, but that was an open label, will not be included in this data. In most cases it showed up after the first dose or the second dose. Its the first or the second. In one case it was the 3rd dose, and in one case it was the 4th dose. Overwhelming number on the first and second. They automatically look at 6 weeks after each dose. They know when to capture it. [T]hat is when it is optimum, at best. This is an easy thing for the doctors to see, its really obvious. It takes some weeks to resolve. In some cases a couple of months. Some were treated with steroids. The degree of it, and the presence or absence of symptoms are very variable. In this

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		current P2 trial, only 4 cases got symptoms. One had abnormal gait and confusion. Other hand funny visual symptoms, wavy lines. Other had confusion. Third had headache, lethargy and confusion. Its variable. The symptoms go away once you stop dosing. Some of the MMSE actually improved with the symptoms. One went from 23 to 28. 17 to 23. 10 to 8. 17 to 23. 26-27. 25-26. [T]here is some random variation in there. Some of them went strikingly up. Some cases of vasogenic edema did show a decline in their MMSE." "If you go back to the numbers again, in the carrier group, they had roughly 40 patients that were getting drug, 40 placebo? In the treated carrier group, they had probably 10 cases lost. They had a total of 12 patients with vasogenic edema in the P2 trial. 2 were non carriers. 10 were carriers. That 40 was brought down to 30 because of the 10 with vasogenic edema. They didn't get a full complement of drug. The way the numbers broke down of the 12 with vasogenic edema. 6 got retreated, and 6 did not. Among those who were treated, they were delayed. Did not give them drug until the vasogenic edema was gone. Then started at the lowest dose. Then they went back up to the next dose in line the .5 dose. If they tolerated that they would continue to go up." "If they were at 2 mg/kg they the stop until symptoms cleared. Would never go all the way up to 2. [W]ould stay one [ ] below the dose that caused vasogenic edema. This means that those 10 patients could be seen as being under treated. Presumably 5 were undertreated, and 5 were not treated. Now they are down to 30 of the carriers to compare with 40 of the placebo. If you think of the carrier group they have more beta amyloid load per volume than the carriers. So would imagine you would need greater dose of drug or longer duration than in the non-carrier group. If these people stayed in the trial he would venture to say the results are a lot better than printed here." "The .15mg was at the request of his committee. In the P1 trial there were 3 cases of vasogenic edema. That petrified him because they were worried from the AN1792. he asked that they go down to a lower dose, because they had already started the .5mg dose. That was only because they were skittish. As originally

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		designed it was only a 3 dose trial."
The incidence of vasogenic edema in the Phase II bapi trial was discussed at a July 8, 2008, GLG Innovators Event and would have been heard by numerous analysts.	DX 1116-A, at 1 (Kathryn Lyndon's notes of a July 8, 2008, GLG Innovators Event).	"GLG panel w/ Sid Gilman, Lon Schneider, Paul Solomon" * * * "In particular the relationship btwn vasogenic edema and APOE carrier status had been determined. Carriers are at higher risk. Mechanism of this is very interesting. APOE carriers have dense collection of beta amyloid in their blood vessels, to a much greater degree than non-carriers. He suspect that beta-amyloid is lifted off the wall. This may weaken the wall and allow more fluid to pass. 10:2 carrier vs. non-carrier that were effected. And the higher the dose the more so. This is one reason for looking. And those effected by vasogenic edema, most did not get symptoms, those who did mild/transient. Many dropped out, for various reasons. Those that did not were treated w/ lower doses. So this group was not treated as frequently as the other group, so this could be the reason for difference in the efficacy[.]"
	Tr. at 2154:10-2155:16.	[*Kathryn Lyndon read the above language from DX 1116-A*] 4 Q. Fair to say, Ms. Lyndon, that you can't remember which of 5 the panelists said that, but fair to say that one of the 6 panelists said that in the group because you took it down in 7 your notes, right? 8 A. That's right. 9 Q. Fair to say that this was, again, a room filled with 10 analysts where the panelists were talking publicly about their 11 view of the bapineuzumab Phase II clinical trial, right? 12 A. That's right. 13 Q. Whoever would have said it, the people in the room, 14 including the panelists like Dr. Gilman, would have heard that 15 being said publicly to everyone who was in the room, right? 16 A. That's right.

EVIDENCE	CITATION	TRANSCRIPT/EXHIBIT REFERENCE
	Tr. at 1712:17-1713:20.	17 Q. In fact, isn't it true, Dr. Gilman, that you were among the 18 headliners at a July 8, 2008 GLG innovators meeting that was 19 open to many investment professionals and clients of GLG? 20 A. July 8? Do you recall where that was held? Was that in 21 New York City? 22 Q. Well, do you remember speaking at a GLG innovators meeting 23 on July 8, 2008? 24 A. If it is the one I'm recalling, it was in New York City. 25 Is that the one you are referring to? 1 Q. Well, do you recall being on a panel with two other 2 doctors, Dr. Lon Schneider and Dr. Paul Solomon? 3 A. Yes, that was -- yes, that was in New York, yes, and I do 4 recall that meeting. * * * 17 Q. The question to you was: Do you recall having attending 18 this innovators meeting on July 8, 2008? 19 A. July 8, thank you. 20 Yes, I do.
Dr. Joel Ross publicly stated to investors that vasogenic edema was a side effect of bapi on an April 9, 2008 Piper Jaffray investor call.	DX 540, at 1 (April 10, 2008, Piper Jaffray analyst report discussing an April 9, 2008, Piper Jaffray investor call).	"Our expert noted that there have been reports of patients experiencing symptomatic and asymptomatic vasogenic edema in the ongoing bapineuzumab Phase II trial and that those cases have been manageable."
	Tr. at 2423:16-25.	[*Anthony Cecchini read the above language from DX 540*] 23 Q. And when you refer to "our expert" here, who are you 24 referring to? 25 A. Dr. Ross.
Elan and Wyeth disclosed that vasogenic edema was a	DX 134, at 16 (April 5, 2006, SMC Meeting	"AAB-001-201 Amendment 4: ICF

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side effect of bapi to potential participants in the Phase II trial and their families as part of the process of obtaining informed consent, beginning in December 2007. Dr. Sidney Gilman testified that these individuals were under no confidentiality obligation with respect to such information.	Minutes).	• AAB-001 may cause swelling of the brain in some patients. • Brain swelling may be associated with inflammation or bleeding into the brain. • Clinical symptoms associated with brain swelling may include: altered mental functioning (e.g., increased confusion, disorientation, and hallucinations), difficulty walking, impaired vision, elevated blood pressure, vomiting, headache, and dizziness, or may be associated with no clinical symptoms."
	DX 755, at 11 (Informed Consent Form for bapi, dated December 5, 2007).	"What Are The Side Effects Of Bapineuzumab? * * * Bapineuzumab may cause swelling of the brain in some subjects."
	Tr. at 1601:5-1602:10.	[*Dr. Sidney Gilman read the above language from DX 755*] 1 Q. Is it fair to say that that is a reference to vasogenic 2 edema? 3 A. Yes. 4 Q. And being familiar with the informed consent forms for the 5 patients involved with the Phase II trial, you are aware that 6 there is no confidentiality obligation that the patients or 7 their loved ones are under with respect to anything that 8 they're told about the side effects of bapineuzumab; isn't that 9 right? 10 A. That's right.
Dr. Joel Ross testified that he discussed that vasogenic edema was a side effect of bapi with potential patients in the Phase II trial.	Tr. at 745:20-746:21.	20 Q. If we could turn now to page 11. 21 And, Dr. Ross, included in that is a section on the 22 informed consent form that discusses the side effects of 23 bapineuzumab, correct? 24 A. Yes. 25 Q. And, again, the date of this was December 2007, correct? 1 A. Yes.

EVIDENCE	CITATION	TRANSCRIPT/EXHIBIT REFERENCE
		2 Q. And the informed consent form that you said you went over 3 with every patient -- and not just every patient, you went over 4 it with people that you were recruiting into the trial, 5 correct? Some people wouldn't participate? 6 A. Can you restate that, please? 7 Q. You went over the potential side effects not only with the 8 patients who agreed to participate in the clinical but with the 9 patients who ultimately refused to participate, isn't that 10 right? 11 A. Yes. 12 Q. And what you told those patients and recruits for the 13 trials was that bapineuzumab may cause swelling of the brain in 14 some subjects, is that correct? 15 A. Yes. 16 Q. And you also told them that swelling could be seen as a 17 change on an MRI scan, isn't that right? 18 A. Yes. 19 Q. And this swelling -- we have gone over it before -- is also 20 called vasogenic edema, isn't that right? 21 A. Yes.
Analyst reports publicly discussed that vasogenic edema was a side effect of bapi in the Phase II trial.	DX 748, at 2 (April 20, 2006, A.G. Edwards & Sons analyst report).	"Elan indicates that the cause of the MRI abnormalities remains unknown, but that they appear to be consistent with vasogenic edema – a shift of water from vessels into the brain."
	DX 754, at 1 (October 26, 2007, Natixis Bleichroeder analyst report).	"We infer from this that the interim Phase II revealed that ApoE carriers responded better to low doses of the drug (and are perhaps more susceptible to vasogenic edema)."
	DX 992-A, at 43 (March 5, 2008, Credit Suisse analyst report).	"**Vasogenic Edema seen in the ongoing phase II study** Cases of vasogenic edema have also been observed in the ongoing phase II study, which is looking at doses of AAB-001 lower than 5 mg/kg. All patients displaying this quickly recovered when taken off the drug and were then subsequently re-

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		challenged with a lower dose and treatment continued (in which no further evidence of edema presented itself)." * * * [It] has been said to be more abundant in ApoE4 carriers."
	DX 1143-A, at 1 (May 15, 2008, Caris & Company analyst report).	"Additionally, data from bapineuzumab studies have revealed patients with vasogenic edema (fluid in the brain), which may pose a risk to broad approval."
	DX 1118-A, at 2 (June 25, 2008, Summer Street analyst report).	"The vasogenic edema is less troublesome without neurologic symptoms. Our experts feel the edema is concerning, however unless the patients have clinical symptoms of encephalitis it will not stop bapineuzumab from becoming widely used. The experts are less troubled by the adverse event because the severity of Alzheimer's disease justifies using risky drugs. The FDA is likely to have already considered the risk of vasogenic edema. Our experts believe the FDA has already considered the risk of vasogenic edema and has allowed the phase III studies to proceed."
	DX 1144-A, at 1, 3 (July 21, 2008, Cowen & Company analyst report).	"**Is Safety An Issue?** We expect the vasogenic edema issues will be a concern only in ApoE4 carriers: clinical events data will be key." * * * "BAPINEUZUMAB PHASE II DATA AT ICAD – WHAT WE EXPECT TO SEE" Comparing ApoE4(-) non-carriers and ApoE4(+) carriers with respect to "Safety signals": "Low incidence of vasogenic edemas (approximately 3-4 total)" in ApoE4 non-carriers, as compared to "Higher incidence of vasogenic edema (approximately 15 total)" in ApoE4 carriers.
The information that Dr. Gilman and Dr. Ross allegedly shared concerning vasogenic edema was not material.		
Dr. Enchi Liu testified that the SMC concluded that vasogenic edema was a	Tr. at 935:16-21.	16 Q. With regard to the top bullet, "VE is a manageable clinical 17 side effect," do you know if that was the conclusion of the 18 SMC?

EVIDENCE	CITATION	TRANSCRIPT/EXHIBIT REFERENCE
manageable side effect.		19 A. Yes. 20 Q. Was it? 21 A. Yes, it was.
Dr. Sidney Gilman testified that vasogenic edema was considered a manageable side effect.	Tr. at 1218:5-12.	5 Q. As the drug trial continued, did you continue to have 6 concerns about vasogenic edema? 7 A. Yes, I had concerns about vasogenic edema as the trial 8 continued. 9 Q. Were they -- did those concerns ever decrease as time went 10 on or increase as time went on? 11 A. They decreased as time went on. I began to see it as a 12 manageable side effect.
Dr. Enchi Liu testified that vasogenic edema was often asymptomatic and would typically resolve itself through time.	Tr. at 951:11-952:6.	11 Q. Am I right vasogenic edema was typically something that was 12 only discovered that the patient had by having an MRI scan done 13 of their brain, right? 14 A. Yes. 15 Q. In most cases, the patient and the doctor didn't even know 16 that the patient was afflicted with this condition just based 17 on their interactions, correct? 18 A. That's true, the large majority were asymptomatic. 19 Q. Now -- 20 A. Clinically, that is. Sorry. 21 Q. So, in other words, just so we make sure we all 22 understand -- I know that term, and I think I understand it, 23 but I have been studying this stuff -- so asymptomatic 24 clinically means they didn't show any outward signs of having 25 vasogenic edema, and you needed to do this MRI scan to actually 1 observe that they may have been afflicted with it, correct? 2 A. Yes, that's true. 3 Q. Also, the vasogenic edema resolved itself typically by just 4 having some time go by and then the swelling associated with 5 the edema would go down, right?

EVIDENCE	CITATION	TRANSCRIPT/EXHIBIT REFERENCE
		6 A. That's correct.
Dr. Thomas Wisniewski testified that information that Dr. Gilman allegedly shared regarding the number of patients who experienced vasogenic edema was not meaningful.	Tr. at 2721:2-11.	2 Q. Does it matter that the ICAD draft presentation identifies 3 the specific number of patients who experienced vasogenic 4 edema? 5 A. No. One would have assumed that this complication would be 6 occurring in a minority of the patients since the press release 7 already indicated that they were proceeding to Phase III 8 clinical trial. So if vasogenic edema was found in a majority 9 of treated patients or more than a relatively small percentage 10 of patients, then the Phase II would not proceed to Phase III 11 testing; it would not be designated as safe.
Dr. Ross testified that the information that he allegedly shared regarding the single case of vasogenic edema that he observed was not statistically significant.	Tr. at 749:2-750:9.	2 Q. And when you run a medical trial, a clinical trial, you 3 understand you don't have to be a statistician to understand 4 the importance of something being statistically significant, 5 right? 6 A. Correct. 7 Q. And you understand that when you run a trial, just one 8 person improving does not tell you anything, does it? 9 A. Correct. 10 Q. Because one person out of a trial of 240, it could just be 11 chance, it could be luck, correct? 12 A. Correct. 13 Q. So even though you're not a statistician, you would agree 14 with me that there has to be some type of what they call 15 statistically significant result to have any meaning, any 16 significance? 17 A. Correct. 18 Q. You don't shut down your clinic if there is one person who 19 has a side effect, correct? 20 A. It depends on the side effect. 21 Q. Well, for instance, the one case of vasogenic edema, you 22 didn't shut down your clinic then, did you?

EVIDENCE	CITATION	TRANSCRIPT/EXHIBIT REFERENCE
		23 A. The study was not halted for the reasons we discussed. We 24 thought it was an acceptable side effect for a fatal disease. 25 Q. And it is important to have the statistical significance 1 because you need to know how different people are going to 2 react to different drugs, isn't that right? 3 A. Yes. 4 Q. Different people have different characteristics, and so 5 drugs could affect them differently, isn't that correct? 6 A. Yes. 7 Q. And so that's why you test on a large number of people, 8 isn't that right? 9 A. Yes.
Elan publicly discussed the hypothesis that vasogenic edema was an indicator of bapi's efficacy in a October 25, 2007, earnings conference call.	DX 173-A, at 12 (Transcript of October 25, 2007, Elan Q3 Earnings Call). *See also* DX 173 (Audio of October 25, 2007, Elan Q3 Earnings Call).	"Lars Ekman – Elan Corporation, PLC – EVP, President of Global R&D, Head of Nuerodegeneration Franchise Vasogenic edema is, as I've said many time before, it's a largely asymptomatic and transient MRI finding. We have learned that it is more abundant in APOE for carriers. That has in affluenced the design of the trial so that we can minimize this event. We do not know yet, if that is an adverse event, or if it's an event that we're actually striving for, but we have at this point said, let's try to maximize that event, and therefore we would only do one dose in those patients."
The hypothesis that vasogenic edema was an indicator of bapi's efficacy was publicly discussed in a March 5, 2008, Credit Suisse analyst report.	DX 992-A, at 43-44 (March 5, 2008, Credit Suisse analyst report).	"***Vasogenic edema may point towards efficacy*** Vasogenic edema is a specific form of edema caused by increased interstitial fluid accumulating in the brain. We believe that the cases of vasogenic edema seen in clinical testing of AAB-001 maybe an unfortunate but important marker of the drug's efficacy. On vasogenic edema, Elan made the following intriguing comments:- '*We do not know yet, if that is an adverse event, or if it's an event that we're actually striving for*.' **Lars Ekman—Elan. Global R&D. Q3'07 Results**"
Dr. Enchi Liu testified that the hypothesis that	Tr. at 956:4-22.	4 Q. Fair. Are you aware that at this time there was discussion 5 among the scientific community about whether in fact this

EVIDENCE	CITATION	TRANSCRIPT/EXHIBIT REFERENCE
vasogenic edema was an indicator of bapi's efficacy was not a proven fact.		6 observed side effect in Phase I of vasogenic edema might 7 actually be a good sign that bapineuzumab was working the way 8 it was supposed to work, but no one was really sure if that was 9 the case or not? 10 A. You know, I don't recall specific discussions around that, 11 but I think evolving, the thinking is this could mean that 12 bapineuzumab had a biological effect. 13 Q. Right. And you could hypothesize that from the fact that 14 you saw vasogenic edema occurring in some of these Phase I 15 patients, right? 16 A. Hypothesize, yeah. 17 Q. But, certainly, I think you said in your direct, certainly 18 no one really knew if that was the case or not, right? 19 A. Knew whether it was the case or not what? 20 Q. Knew whether it was the case or not that, in fact, 21 bapineuzumab was working or not, right? 22 A. No, we don't know.
Dr. Sidney Gilman testified that the hypothesis that vasogenic edema was an indicator of bapi's efficacy was not a proven fact.	Tr. at 1715:8-1716:14.	8 Q. And now you recall, right, Dr. Gilman, that you shared with 9 the financial analysts in this meeting your thought that the 10 fact that you were seeing more vasogenic edema in carriers of 11 that ApoE4 gene meant or might mean that that bapineuzumab was 12 working to remove the beta-amyloid from the walls of the blood 13 vessels in the brain? 14 A. I presented this as a hypothesis, sir, not as a fact. 15 Q. And, in fact, it was a hypothesis, correct, Dr. Gilman? 16 A. Yes, that is my hypothesis. 17 Q. In fact, even today it is not a fact that it actually -- 18 bapineuzumab actually works, correct? 19 A. By "works," do you mean -- 20 THE COURT: You are talking over each other so you've 21 got to let him finish his answer. 22 Finish your answer.

EVIDENCE	CITATION	TRANSCRIPT/EXHIBIT REFERENCE
		23 MR. STRASSBERG: I think it might be the question was 24 difficult, your Honor. So I'm happy for him to finish but I am 25 happy to rephrase it. 1 THE COURT: All right. So rephrase the question. 2 BY MR. STRASSBERG: 3 Q. So at the time, as you were seeing the results of vasogenic 4 edema in the Phase II trial, it was your hypothesis, right, 5 that that might be a signal that bapineuzumab was removing the 6 beta-amyloid plaques from the blood vessels of the brain? 7 A. Yes, sir. 8 Q. And that remained the hypothesis throughout this time 9 period in 2008, correct? 10 A. Yes. 11 Q. And there was nothing about the ultimate trial results of 12 the Phase II trial from bapineuzumab that confirmed whether 13 that hypothesis was actually occurring or not, correct? 14 A. That's right.
The hypothesis that vasogenic edema was an indicator of bapi's efficacy was ultimately disproved because, as Dr. Sidney Gilman testified, there are no drugs on the market that can cure Alzheimer's disease.	Tr. at 1526:23-1527:4.	23 Q. And am I right, Dr. Gilman, that there are a number of 24 drugs on the market now, and there were then, that could treat 25 the symptoms of Alzheimer's, right? 1 A. Yes. 2 Q. But there is no drug then and no drug now that can actually 3 help to cure the disease, right? 4 A. That's right.
The hypothesis that vasogenic edema was an indicator of bapi's efficacy was ultimately disproved because, as Dr. Allison Hulme testified, there are	Tr. at 267:24-269:10.	24 Q. Is there a cure to date for Alzheimer's disease? 25 A. No, there is nothing that can stop the progression or cure 1 Alzheimer's. 2 Q. Are there any effective treatments on the market to date? 3 A. There are some treatments that are on the market, and they 4 treat the symptoms that a patient may exhibit for Alzheimer's,

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no drugs available that can stop the progression of Alzheimer's disease.		5 but they don't actually stop the progression of the disease. 6 Q. When you say stop the progression, you mean what? 7 A. That it can't prevent the patient for continuing to suffer 8 damage to the brain which can interrupt all these processes 9 we've just been mentioning, like thinking and behavioral 10 changes, so they continue to progress. 11 Q. So, when you say there are drugs though that can treat the 12 symptoms of the disease, can you give an example? 13 A. Aricept, which will help with some of the mental confusion 14 is one that is more commonly known. 15 Q. Does Alzheimer's disease when it strikes cause any physical 16 effects in the brain itself that one could examine? 17 A. Yes, it's characterized by the buildup of large -- or 18 buildup of sticky protein called amyloid which will interfere 19 with the communication between the nerve cells and that is 20 what's resulting in the symptomatology such as the confusion, 21 and memory loss, etc. and can actually cause the death of those 22 nerve cells in the brain as well. 23 Q. Do people sometimes refer to this protein amyloid substance 24 as a plaque? 25 A. Yes, they do. The buildup and the deposition of this 1 protein, this sticky toxic protein in the brain is often 2 referred to as a plaque by many people. 3 Q. Is it known at this point whether removing the plaque 4 treats the disease or is that something that's still under 5 study? 6 A. It's under study, but there are animal models that have 7 demonstrated that removal of the plaque can result in 8 improvement in the behavioral symptoms in mice, for example. 9 Q. But not in humans yet? 10 A. Right, not in humans.
(b) ENROLLMENT DATA		

EVIDENCE	CITATION	TRANSCRIPT/EXHIBIT REFERENCE
The information that Dr. Ross allegedly shared concerning enrollment data was public.		
The government website www.clinicaltrials.gov posted enrollment information for the Phase II bapi trial on October 3, 2006.	DX 652, at 1-2 (www.clinicaltrials.gov website, updated October 3, 2006).	"**Brief title** AAB-001 in Patients With Mild to Moderate Alzheimer's Disease" * * * "**Expected enrollment** 240"
Elan and Wyeth publicly discussed the enrollment figures for the Phase II bapi trial in a May 21, 2007, press release.	DX 1079, at 2 (Elan and Wyeth's May 21, 2007, press release).	"The first Phase 2 trial is a randomized, double-blind, placebo controlled, multiple ascending dose study of 4 cohorts of the approximately 240 total patients with mild to moderate Alzheimer's disease."
Elan and Wyeth publicly discussed the enrollment figures for the Phase II bapi trial in a June 17, 2008 press release.	GX 10, at 2 (Elan's and Wyeth's June 17, 2008, press release).	"The study was designed to enroll approximately 240 participants at 29 sites in the United States. The study tested four doses of bapineuzumab (0.15 mg/kg, 0.5 mg/kg, 1.0 mg/kg and 2.0 mg/kg) with approximately 60 patients in each dose cohort. Patients were randomized on an 8:7 ratio to receive bapineuzumab or placebo, resulting in approximately 32 participants receiving bapineuzumab and 28 participants receiving placebo in each dose group."
Dr. Joel Ross publicly shared the total enrollment figures in the Phase II bapi trial with investors, during an April 9, 2008, Piper Jaffray investor call.	DX 540, at 1 (April 10, 2008, Piper Jaffray analyst report discussing April 9, 2008, Piper Jaffray investor call).	"In the study, 240 patients were randomized in 4 dosing cohorts with a treatment duration of 18 months."
	Tr. at 2423:16-2424:4.	[*Anthony Cecchini read from DX 540*] 23 Q. And when you refer to "our expert" here, who are you 24 referring to? 25 A. Dr. Ross. 1 Q. Is it fair to say that everywhere in this analyst report 2 where you refer to our expert, our guest physician, or our

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		3 consultant, you are referring to Dr. Ross? 4 A. Yes.
The information that Dr. Ross allegedly shared concerning enrollment data was not material.		
Dr. Joel Ross testified that enrollment figures say nothing about bapi's safety or efficacy.	Tr. at 755:15-756:1.	15 Q. And just for what it's worth, enrollment data doesn't tell 16 you if the drug is going to save any lives, does it? 17 A. No. 18 Q. Enrollment data doesn't tell you if the drug is going to be 19 approved by the FDA? 20 A. No. 21 Q. Enrollment data doesn't tell you if the drug is safe, 22 obviously? 23 A. No. 24 Q. Enrollment data doesn't tell you if the drug is effective 25 in any way, does it? 1 A. No.
Dr. Ross was involved in the Phase II bapi trial at only one of 29 different clinical investigation cites.	GX 10, at 2 (Elan's and Wyeth's June 17, 2008, press release).	"The Phase 2 trial was . . . . designed to enroll approximately 240 participants at 29 sites in the United States."
(c) DROPOUT RATES		
The information that Dr. Gilman allegedly shared concerning dropout rates was public.		
Dr. Sidney Gilman publicly shared dropout information from the Phase II bapi trial with investors during an April 15, 2008, roundtable.	DX 759, at 2 (Rene Shen's notes from an April 15, 2008, roundtable with Sid Gilman).	".5mg you will end up having 20 and 20 with drop outs. To show a benefit is very difficult."
Dropout rates in the Phase II bapi trial were discussed at a July 8, 2008, GLG Innovators Event and would have been heard by	DX 1116-A, at 1 (Kathryn Lyndon's notes of the July 8, 2008, GLG Innovators Event).	"And those effected by vasogenic edema, most did not get symptoms, those who did mild/transient. Many dropped out, for various reasons. Those that did not were treated w/ lower doses. So this group was not treated as frequently as the other group, so this could be the reason for difference in the efficacy[.]"

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numerous analysts.	Tr. at 2154:10-2155:16.	[*Kathryn Lyndon read the above language from DX 1116-A*] 4 Q. Fair to say, Ms. Lyndon, that you can't remember which of 5 the panelists said that, but fair to say that one of the 6 panelists said that in the group because you took it down in 7 your notes, right? 8 A. That's right. 9 Q. Fair to say that this was, again, a room filled with 10 analysts where the panelists were talking publicly about their 11 view of the bapineuzumab Phase II clinical trial, right? 12 A. That's right. 13 Q. Whoever would have said it, the people in the room, 14 including the panelists like Dr. Gilman, would have heard that 15 being said publicly to everyone who was in the room, right? 16 A. That's right.
	Tr. at 1712:17-1713:20.	17 Q. In fact, isn't it true, Dr. Gilman, that you were among the 18 headliners at a July 8, 2008 GLG innovators meeting that was 19 open to many investment professionals and clients of GLG? 20 A. July 8? Do you recall where that was held? Was that in 21 New York City? 22 Q. Well, do you remember speaking at a GLG innovators meeting 23 on July 8, 2008? 24 A. If it is the one I'm recalling, it was in New York City. 25 Is that the one you are referring to? 1 Q. Well, do you recall being on a panel with two other 2 doctors, Dr. Lon Schneider and Dr. Paul Solomon? 3 A. Yes, that was -- yes, that was in New York, yes, and I do 4 recall that meeting. * * * 17 Q. The question to you was: Do you recall having attending 18 this innovators meeting on July 8, 2008? 19 A. July 8, thank you.

EVIDENCE	CITATION	TRANSCRIPT/EXHIBIT REFERENCE
		20 Yes, I do.
Dr. Sidney Gilman discussed dropout rates in the Phase II bapi trial with David Munno on May 1, 2008.	DX 839, at 1 (May 1, 2008, e-mail from Dr. Sidney Gilman to David Munno).	"Dear Dr. Munno: I just discovered today that I gave you an incorrect total number of cases that completed study in the Phase II trial of Bapineuzumab. The reason is that the sponsor made the mistake, and yesterday I reviewed the data, corrected the total and informed the sponsor, who agreed that the sponsor had made an error. I quoted a total number of cases that completed all 6 immunizations as 126 (out of the 234 who began the trial). The number of completers should be 164 and not 126."
Dr. Sidney Gilman discussed dropout rates in the Phase II bapi trial during a consultation with Rene Shen on June 17, 2008.	DX 761, at 1 (Rene Shen's notes from his June 17, 2008, consultation with Sid Gilman).	"Thinks that had 80 completers with carriers, and 80 with the non-carriers. So really 40 drug vs. 40 placebo in each group." * * * "Vasogenic edema would have caused 5-6 of the dropouts. Could cause the other 6 to be undertreated. It could be a delay of some months. Gradual uptitration of the dose." * * * "The number of recruited patients was 8:7. as originally recruited was 1:1. the 8 to 7 was an assumptions about drop outs. Think that they were assuming there were more dropouts with the placebo group. Often the trial in 18 month trials. Not sure if this is the case here." * * * "Thinks that for the carriers- when you drop out those cases that had complications. He bets that they are going to be very close to the non-carriers. Even though the numbers are small. You are going down from 40 down to 30 that are going to drug treated. thinks that there is a drug effect in the carriers."
The information that Dr. Gilman allegedly shared concerning dropout rates was not material.		
Dr. Enchi Liu testified that dropouts occur in clinical drug trials for a variety of reasons.	Tr. at 915:6-9.	6 Q. And for what reasons did dropouts and study attrition occur 7 in this trial? 8 A. There are a variety of reasons, the major one of which was 9 adverse events that may or may not be related to study drugs.

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Dr. Allison Hulme testified that the dropout rate for the Phase II bapi trial was reasonable.	Tr. at 415:11-20.	11 Q. Ms. Hulme, you were asked questions about the dropout rates 12 by the prosecutor just now. Am I correct that there was no 13 problem with a high dropout rate in connection with the Phase 14 II study of bapineuzumab, right? 15 A. Correct, for an 18-month study, it was reasonable in both 16 groups. 17 Q. Fair to say that the dropout rate for the bapineuzumab 18 Phase II study was consistent with historical rates for these 19 kinds of trials? 20 A. That is correct.
Dr. Allison Hulme testified that dropout rates are not indicative of the efficacy of a drug.	Tr. at 357:3-24.	3 Q. Isn't it true, however, that the information on dropouts or 4 the dropout rate in a double-blinded study was not considered 5 to be a particularly useful metric because you can't tell which 6 patients dropped out that were taking the drug or which 7 patients dropped out that were on the placebo? 8 MR. DEVLIN-BROWN: Objection. 9 THE COURT: Grounds. 10 MR. DEVLIN-BROWN: Beyond the scope. 11 THE COURT: Overruled. 12 A. So the question, I believe, was the dropouts, we wouldn't 13 know whether they were randomized or were receiving active 14 versus placebo and therefore that would not tell you anything 15 about the effectiveness of the drug? 16 Q. Yes, I think you said it perhaps better than I did. 17 A. Then in a double-blind situation, that withdrawal rate 18 should not -- you can guess whether it's active or was placebo, 19 but it's speculative. 20 Q. So, is it fair to say it really doesn't give you any useful 21 guidance as to whether the drug is being effective or not? 22 A. You could be completely wrong, so -- you can guess, but you 23 may also get it wrong. I had that example happen to me in the

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		24 past.
Dr. Thomas Wisniewski testified that dropout rates of 20-30% are common in clinical trials, and such rates are not indicative of the safety or efficacy of a drug.	Tr. at 2717:19-25.	19 Q. What about the rest of the information in those boxes on 20 slide four, sort of middle of the page? Do they tell us 21 anything about the safety or efficacy of bapineuzumab? 22 A. No, they do not. In clinical trials, as one can imagine, 23 patients drop out for lots of different reasons, and having a 24 dropout rate of 20 to 30 percent, particularly in a fairly long 25 clinical trial, is a very common circumstance.
Dr. Allison Hulme testified that the dropout rate for the Phase II bapi trial would not be significant to investors and was not meaningful to her, because it did not distinguish between patients who received bapi and patients who received a placebo.	Tr. at 406:4-9.	4 When you testified on cross-examination that the 5 dropout rate was not particularly important to you, do you know 6 one way or the other whether the dropout rate could be 7 significant to investors? 8 A. I don't see how when they don't know whether it's 9 bapineuzumab or placebo.
	Tr. at 402:23-403:5.	23 Q. Do you recall being asked some questions on 24 cross-examination about the dropouts in the trial, how many 25 people had left the trial at a particular time? 1 A. Yes, I do. 2 Q. And you would recall being asked whether that information 3 was meaningful to you, and you essentially said no? 4 A. That's correct, because you don't know which group is 5 active and which group is placebo.
Dr. Enchi Liu testified that the dropout rates indicated in slides presented at the March 15, 2008, SMC meeting did not distinguish between patients that received bapi and patients that received a placebo.	Tr. at 915:10-25.	10 THE COURT: I had a question. Would the column that's 11 "Number of patients active: Placebo," does this indicate that 12 61 received the drugs and presumably another 61 received the 13 placebo, or does it indicate that -- or does it indicate 14 something different? 15 THE WITNESS: No. It was a total of 61. So 16 approximately 30 would have gotten the active bapineuzumab and 17 approximately 30 would have gotten the placebo. But at this 18 point the study is blinded so we don't know exactly.

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		19 THE COURT: Right. So the dropoff that is shown in 1 20 through 6 reflects both patients who had received the active 21 drug, the active ingredient, and patients -- 22 THE WITNESS: Right. At this point we don't know -- 23 THE COURT: -- who received a placebo, and you don't 24 know which? 25 THE WITNESS: Yes. So -- mm-hmm.
Dr. Thomas Wisniewski testified that high dropout rates may indicate that there is something wrong with the design of the drug trial.	Tr. at 2797:8-2798:11.	8 Q. And if in the middle of a trial you learn the dropout rates 9 are exceeding the normal, it could be because the drug is 10 causing safety problems or lacks efficacy, right? 11 A. That's true. But in those circumstances there would be a 12 difference in the dropout rate between the treated versus the 13 placebo. 14 Q. But if all you knew while the trial was ongoing was that 15 the total dropout rate was hypothetically 50 percent, you could 16 take that fact and infer that there could potentially be an 17 issue with the safety or efficacy of the drug that's causing a 18 higher than normal dropout? 19 A. So it would depend on if the data was -- if it was stated 20 that there was a 50-percent dropout rate among the treated 21 patients. That would be my conclusion. If it was just stated 22 that there was ace 50 percent dropout rate in the clinical 23 trial, then that is sort of neutral because you don't know if 24 it is equal among the placebo and in the treated group, and, 25 for example, that may happen in many neurological diseases, for 1 example, because the patients die because it is such a dire 2 condition, for example. 3 Q. But with Alzheimer's Disease if the number for you isn't 4 50 percent, there is some percent where you can infer that 5 there is something that's not going well with the medication, 6 right? Like an 80 percent dropout rate is going to signal 7 something, isn't it?

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		8 A. So it would signal that there is something wrong with the 9 design of the trial but not -- because if it is 80 percent 10 dropout rate and it's in the placebo and treated, then there is 11 something wrong with the trial.
(d) LACK OF DOSE RESPONSE		
The information that Dr. Gilman allegedly shared concerning the lack of dose response was public.		
A July 11, 2008, Brean Murray analyst report publicly discussed the lack of dose response observed in bapi.	DX 9, at 6-7 (July 11, 2008, Brean Murray analyst report).	"Phase I Trial Results Not Encouraging -- Lack of Dose Response and Severe Side Effects" "We are not encouraged by the lack of a dose response (see Exhibit 4) or by the safety signals at the 5 mg/kg dose." Exhibit 4 shows the lack of dose response as seen in Wyeth-generated graphs.
A March 5, 2008, Credit Suisse analyst report publicly discussed the lack of dose response observed in bapi.	DX 992-A, at 42-43 (March 5, 2008, Credit Suisse analyst report).	"The study showed that the drug was generally well tolerated and in six patients that received one dose of 1.5mg/ Kg bapineuzumab there was a statistically significant benefit in cognitive function on the MMSE scale after four months when compared to the eight placebo patients." * * * "Patients treated with the 5 mg / kg dose failed to show any cognitive benefit when compared with placebo." Figure 48 shows the lack of dose response as seen in Wyeth-generated graphs.
The information that Dr. Gilman allegedly shared concerning the lack of dose response was not material.		
Dr. Allison Hulme publicly stated that the individual dose cohorts (*i.e.*, sample sizes) in the Phase II bapi trial were very small, which rendered the lack of dose response data	DX 177-A, at 7-8 (Transcript of July 29, 2008, Elan and Wyeth conference call). *See also* DX 177 (Audio of July 29, 2008, Elan	Allison Hulme (EVP & Head of Global Development, Elan Corporation, PLC): "I'm going to first start off by reminding us that in the individual dose cohorts that, we have we do have some very small numbers, when we split that patient population into carriers and non-carriers." * * * Allison Hulme (EVP & Head of Global Development, Elan Corporation, PLC): ". .

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inconclusive, during a July 29, 2008, Elan and Wyeth conference call.	and Wyeth conference call).	. I think, again, I'm going to stress that this was a dose cohort study so we have the four dose cohort fill and it really wasn't – these patients didn't come in on those doses and then get a parallel group design into randomizing them. It's very difficult with the numbers that we've got in each of those dose cohorts to really pay a huge amount of attention to individual groups and how they respond on the various outcome measures. That's why we really focus back to the all dose groups, particularly when we go into those sub populations of non-carriers and carriers. Where we're really reassured is that when we look at that total patient population, both the MITT analysis where we saw the trends on the ADAS-cog and the NTB, and then when we look at those patients that got the drug as we designed it to do and we look at all of the doses versus all of the placebo in that population, you see very robust data in terms of changes in both ADAS-cog, NTB, DAD and CDR sum of boxes. So we're not going to be swayed by one individual dose cohort that doesn't behave typical with the other dose cohorts. We combine them into all doses."
Dr. Sidney Gilman testified that the lack of dose response did not change his positive view of bapi's effectiveness after the Phase II trial.	Tr. at 1422:6-1423:5.	6 Q. I think you mentioned you had two principal concerns. What 7 was the second? 8 A. That was the first. First, that there may be excessive 9 carrier decline in the non-carrier group. 10 Concern number two was that there was no dose 11 response. And what I mean by dose response is the following: 12 The best example I can give you is that if you have a headache 13 and you take one aspirin, you get a little bit of relief. If 14 you take two aspirin, you get a little better relief. In other 15 words, there's a dose response. Take three aspirin, you get 16 even more relief. You start getting ringing in your ears; of 17 course, you get a side effect. Generally, most -- not most, 18 but many -- drugs show a better response as you increase the 19 dose. 20 Q. What did the results show in this trial? 21 A. Here, in this trial there was no dose response. It didn't 22 show up at all. So that was the second concern. Not a killer,

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		23 not -- it didn't mean that the drug doesn't work. It was just 24 a concern. 25 Q. Why was it a concern? 1 A. Well, it's a concern because one wants to see evidence that 2 the drug actually works. That would be one way to show that it 3 works, so there were two things about the trial that concerned 4 me a little bit. So, that's all. Otherwise it was marvelous. 5 It was just so exciting
	Tr. at 1455:13-1456:11.	13 Q. Do you recall any details about the July 19 conversation 14 when he was in your office or no? 15 A. I do recall going over the completers analysis for the 16 non-carriers which we described last time in which the placebo 17 group dropped 14 on the scale for the ADAS-cog. I recall 18 showing the carrier group which -- in which the placebo group 19 dropped much less around eight or so. I recall showing him the 20 lack of a dose response. I recall discussion in response to 21 his questions the lack of dose response, and my overall view 22 of -- he asked my view of the results, and my response was, 23 well, these are -- these two items, the marked drop of the 24 placebo group and the lack of a dose response are relative 25 concerns; not huge concerns. I still was very excited about 1 the results. 2 I think they're the first results showing a treatment 3 that is effective, that appears to be effective, at least in 4 the Phase II study. The lack of effect in the carrier group is 5 of perhaps a little concern also but it is still a favorable 6 trend, so I think these are certainly results worthy of Phase 7 III, another larger trial. I'm very excited about the results. 8 Q. The last few sentences you started with "I think." Were 9 those just things you were thinking or were you communicating 10 that to Mr. Martoma? 11 A. I was communicating them.

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Dr. Ross testified that drugs may follow a dose response curve where lower doses may be more effective than higher doses such that the effectiveness of a drug may decrease as the dose increases without indicating that the drug overall was ineffective.	Tr. at 773:18-775:12.	18 Q. But isn't it true that some medications actually have less 19 effect as the dose goes up? 20 A. Possibly. 21 Q. Isn't that concept called hormesis? 22 A. What's the term? 23 Q. Hormesis? 24 A. Not familiar with the term. 25 Q. The hormetic dose response is, in essence, where you 1 increase the dose of a drug, and it's almost like an upside 2 down horseshoe. Are you familiar with that? 3 A. There are what's called dose response curves, some of which 4 have higher doses, give you better results, and sometimes a 5 middle dose is a more effective dose; but I'm not familiar with 6 that term you mentioned. * * * 5 Q. But you'll agree with me then, for some drugs, as you 6 increase the dose, actually the effectiveness of the drug may 7 actually decrease? 8 A. It may happen, but I'm not familiar with which ones. 9 Q. So, when you talked about the dose effect response in these 10 drug trials and talking about how the stepladder would have to 11 increase, that's not always the case for all drugs, is it? 12 A. May not be.
Dr. Thomas Wisniewski testified that one would not expect a clear dose response curve in the Phase II bapi trial.	Tr. at 2737:25-2738:14.	25 Q. Now, Dr. Wisniewski, there has been some testimony in the 1 case regarding dose response or dose effect. 2 Looking at slide 16 of the draft ICAD presentation, 3 what do these graphs tell us, if anything, about whether or not 4 bapineuzumab shows a dose response? 5 A. So the data here does not give any clear indication of a 6 dose response. However, in the setting where we know that 7 there's toxicity at a higher dose of the bapineuzumab, we --

EVIDENCE	CITATION	TRANSCRIPT/EXHIBIT REFERENCE
		8 certainly one would not expect a clear dose response curve. 9 There is some suggestion in the presented data here that at 10 lower doses, the two lower doses, there may be some dose 11 response between the .15 and the .5 doses, but then at the 12 higher dose that is not at all evident. So -- but then again 13 at the higher doses we know that vasogenic edema is an issue, 14 so that could cloud the picture.
Dr. Thomas Wisniewski testified that, in order to determine whether bapi actually had a dose response, a separate clinical trial would have been required.	Tr. at 2738:15-2739:6.	15 Q. If you wanted to test for dose response, is there anything 16 you could do to attempt to see if bapineuzumab actually had a 17 dose response? 18 A. Certainly. So that would require, in fact, another 19 clinical trial where there is less of a spread of the doses 20 being given. So it's possible that, for example, if a clinical 21 trial was done at .15, .5, and .75 milligrams per kilo, then a 22 dose response would be seen. It's very common for 23 pharmaceutical agents to be effective at lower and middle doses 24 but as you go to higher doses you have evidence of toxicity. 25 So, therefore, one wouldn't expect to see a linear 1 dose response curve if you go to toxic levels, where you're 2 having significant toxicity. In this case, vasogenic edema, it 3 would be more likes a dome-shaped response curve. This sort of 4 hermetic response for medications is well known in other 5 Alzheimer's drugs and it is a common phenomena 6 pharmacologically.
(e) PLACEBO RATE OF DECLINE IN ApoE4 NON-CARRIERS		
The information that Dr. Gilman allegedly shared concerning the placebo rate of decline in ApoE4 non-carriers was not material.		
Dr. Sidney Gilman publicly stated on an investor call following the ICAD presentation on July 29, 2008, that the placebo rate of decline in ApoE4 non-	DX 177-A, at 9-10 (Transcript of July 29, 2008, Elan and Wyeth conference call). *See also* DX 177 (Audio	Ian Sanderson (Analyst, Cowen and Company): "[I]n the non-carrier group, the placebo group cohort or placebo groups showed an 11 point decline in ADAS-cog, which is quite a bit higher than the decline we've seen in other 18 month studies. Can you explain why that might be? And similarly, why that differed so significantly or differed so much from the placebo group decline we say in the carriers?"

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carriers was not unusual and was within the standard of what was seen.	of July 29, 2008, Elan and Wyeth conference call).	* * * Sid Gilman: "The study was not initially designed to compare carriers versus non-carriers. This was done as a post hoc evaluation. And therefore it's not surprising that one might see differences between these two groups and the degree to which they descended at 18 months. Moreover, as Dr. Black just indicated, a number of studies including the [cysteine] study done by the Alzheimer's Disease Cooperative Study, showed an average decline of about 6 ADAS-cog, plus or minus 7, deviation of 7. One could easily get down to 13. This is not unusual. It's within the standard of what we're seeing."
Dr. Ron Black, Assistant Vice President of Neuroscience at Wyeth Research, also publicly stated on the July 29, 2008, investor call that the placebo rate of decline in ApoE4 non-carriers was not unusual.	DX 177-A, at 10 (Transcript of July 29, 2008, Elan and Wyeth conference call). *See also* DX 177 (Audio of July 29, 2008, Elan and Wyeth conference call).	Ron Black (Assistant VP Neuroscience, Wyeth Research): "I don't really think that for this 18 month study that the deterioration in either of the groups is very much out of line and unexpected. We just saw a presentation from another sponsor that showed an 8 point decline in an 18 month study and that study was restricted to only mild patients on an ADAS-cog will typically decline less than a mild to moderate population. So I don't agree that this is an unusual decline in the placebo population."
Dr. Thomas Wisniewski testified that the placebo rate of decline in ApoE4 non-carriers was within the standard range for clinical trials and, as a result, such information was not statistically significant.	Tr. at 2744:12-21.	12 Q. OK. And are you aware of the range of variation that can 13 occur when looking at the decline in placebo groups involved in 14 Alzheimer's trials? 15 A. So, there certainly can be quite a wide range, and the 16 range shown here is certainly within the variation that one has 17 from clinical trial to clinical trial and patient group to 18 patient group. 19 Q. If the variation is within a range, can you draw any 20 statistically significant finding from that variation? 21 A. No, you cannot.
Dr. Sidney Gilman testified that the placebo rate of decline in ApoE4 non-	Tr. at 1421:19-1422:5.	19 Q. What was the significance of that to you? 20 A. So, that made it look as if the reason the non-carriers 21 were significant was that the placebo group was responsible for

EVIDENCE	CITATION	TRANSCRIPT/EXHIBIT REFERENCE
carriers did not change his positive view of bapi's effectiveness after the Phase II trial.		22 it. In other words, it was maybe not so much the drug as much 23 as the placebo group declined. That's a possibility and that's 24 a bit of a concern. 25 Q. Was it a sort of deal breaker to you – 1 A. No, it was not a deal breaker. No. It was a clinical 2 trial, and in a clinical trial, you recruit patients, and maybe 3 that just happens to be the way that the non-carriers decline 4 when you look at the placebo group. The trial is what it is, 5 but that was a concern.
(e) BAPI PHASE II TRIAL DATA TO BE PRESENTED AT ICAD		
The information that Dr. Gilman and Dr. Ross allegedly shared concerning the Phase II bapi trial data was not material.		
Dr. Joel Ross testified that the Phase II bapi trial results were no different from the results disclosed in Elan's and Wyeth's June 17, 2008, press release.	Tr. at 714:4-25.	4 Q. What did Dr. Sperling say in response to your question? 5 A. She had told me that, no, the drug did not work at all in 6 terms of the primary outcome. Basically, the drug failed. 7 Q. What did you do after the efficacy presentation was over? 8 A. I departed the meeting. 9 Q. Where did you go? 10 A. I had an appointment with Mr. Martoma. * * * 19 Q. When you got to the lobby, what happened? 20 A. We had set a meeting. We greeted each other. We exchanged 21 greetings, and he asked basically how the meeting was. 22 Q. And what did you say? 23 A. I responded. I said the meeting showed that the results 24 were no different than the June 17, the drug failed to reach 25 efficacy as indicated on June 17, 2008 press report.
Dr. Thomas Wisniewski testified that the there was no meaningful difference between the data in the draft ICAD presentation	Tr. at 2700:15-2701:6.	15 Q. Now, Dr. Wisniewski, have you reviewed what's been 16 previously received into evidence in this case as Government 17 Exhibit 11A, which is a July 17, 2008 draft version of the 18 PowerPoint that was presented at the ICAD conference? 19 A. Yes.

EVIDENCE	CITATION	TRANSCRIPT/EXHIBIT REFERENCE
that Dr. Gilman allegedly shared with Mr. Martoma and the information in Elan's and Wyeth's June 17, 2008, press release.		20 Q. Have you compared the draft PowerPoint that was dated on 21 July 17 of 2008 against the press release Government Exhibit 22 10? 23 A. Yes, I have. 24 Q. In your opinion, did the full results that were reflected 25 in the PowerPoint slides of the draft PowerPoint Government 1 Exhibit 11A, provide any meaningfully different information 2 from that contained in the June 17 press release, Government 3 Exhibit 10? 4 A. It was my opinion that there was no meaningful difference 5 in the data from the press release to the draft ICAD 6 presentation.
	Tr. at 2756:16-23.	16 Q. Having gone through the entire draft ICAD PowerPoint 17 presentation slide by slide, what is your opinion as to whether 18 there is any meaningful differences between the information in 19 the ICAD presentation Government Exhibit 11A and the 20 information in the June press release, Government Exhibit 10? 21 A. That there are no substantive differences between the two. 22 The draft ICAD gives more detail and graphic representation of 23 what's written verbally in the press release.
Dr. Thomas Wisniewski testified that the there was no meaningful difference between the final presentation that was shown at ICAD and Elan's and Wyeth's June 17, 2008, press release.	Tr. at 2701:7-18.	7 Q. Have you had also an opportunity to review the final 8 PowerPoint presentation that was shown at ICAD which has been 9 admitted into evidence in this case as Government Exhibit 19A? 10 A. Yes, I reviewed that also. 11 Q. Have you compared the final PowerPoint, Government Exhibit 12 19A, and the draft PowerPoint, Government Exhibit 11A, and the 13 press release, Government Exhibit 10? 14 A. Yes, I have. 15 Q. What is your opinion as to whether the final PowerPoint 16 presentation revealed any meaningfully different information 17 from that revealed in the press release? 18 A. There was no meaningful difference between those two.